FIG. **6**, the results obtained after 72 hours of treatment with ML00253764 show a significant antiproliferative effect of this compound already at the concentration of 10 nM both in the A-2058 cell line (FIG. **6**A) and the WM-266-4 cell line (FIG. **6**B) with experimental IC_{50}s of 11.1 nM and 33.7 nM, respectively (Table 1). In these studies, the present inventors used as a positive control the compound Vemurafenib, the drug of choice for the treatment of BRAF-mutant melanoma, which is very active in both A-2058 (FIG. **7**A) and WM-266-4 (FIG. **7**B) cell lines, although with significantly higher IC_{50}s (Table 1). In particular, FIG. **7**B shows the antiproliferative effects of Vemurafenib on WM-266-4 cells (IC_{50} 46.6 nM, BRAF V600E), whereas the A-2058 cell line shows less sensitivity to the treatment (IC_{50} 526 nM, BRAF V600D), as shown in FIG. **7**A.

[0067] The present inventors subsequently demonstrated that the MC4R receptor antagonist for use according to the invention is capable of inducing a significant inhibition of the proliferative activity also in the 8305C thyroid carcinoma cell line (FIG. **8**A), and the HT-29 (FIG. **8**B) and Caco-2 (FIG. **8**C) gastrointestinal tumor cell lines. Following treatment with the compound ML00253764 for 72 hours, a significant antiproliferative effect could be seen already at the concentration of 10 nM both in the 8305C cell line (FIG. **8**A) and the HT-29 cell line (FIG. **8**B) with experimental IC_{50}s of 7667 nM and 806.4 nM, respectively (Table 1). The antiproliferative activity of compound ML00253764 in the Caco-2 cell line is shown in FIG. **8**C, with an IC_{50} of 2993 nM (Table 1).

TABLE 1

IC_{50} values of the MC4R antagonist ML00253764 in A-2058 and WM 266-4 human melanoma cell lines, 8305C thyroid carcinoma cell line and HT-29 and Caco-2 gastrointestinal tract tumor cell lines. The drug concentration that reduces cell proliferation by 50% (IC_{50}) compared to controls was calculated by interpolating the mean values of the data obtained in triplicate experiments (at least nine wells for each concentration).

	IC50 [nM] - 72 h	
Melanoma cell lines	ML00253764	Vemurafenib
A-2058	11.1	526
WM 266-4	33.7	46.6
Other cell lines		
8305C	7667	—
HT-29	806.4	—
CaCo-2	2993	—

EXAMPLE 10: SYNERGISTIC THERAPEUTIC EFFECT OF THE COMBINATION OF THE MC4R RECEPTOR ANTAGONIST AND VEMURAFENIB

[0068] In order to verify a potential synergistic effect of the simultaneous treatment by Vemurafenib in combination with the MC4R receptor antagonist for use according to the invention, the present inventors administered these compounds in a fixed 1:10 molar ratio to the two melanoma cell lines. Treatment was carried out for 72 hours. The results of these studies illustrated in the graphs of FIG. **9** show that the simultaneous administration of the combination of ML00253764 and Vemurafenib exerts a marked synergism on the affected cell fractions (Fa) (CI<1) both in A-2058 cells (FIG. **9**A) and WM-266-4 cells (FIG. **9**B).

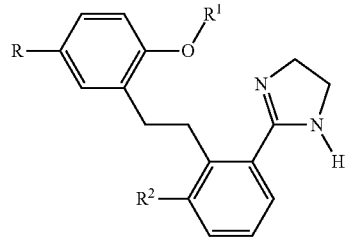
EXAMPLE 11: PRO-APOPTOTIC EFFECT OF THE MC4R RECEPTOR ANTAGONIST FOR USE ACCORDING TO THE INVENTION

[0069] By staining with TO-PROS iodide nuclear marker, visualized with a confocal laser scanning microscope, the present inventors detected a weak fluorescence of normal-sized nuclei of A-2058 melanoma cells (FIG. **10**A) treated with the vehicle alone, indicative of non-apoptotic cells. In contrast, cell samples treated with the compound ML00253764 at a concentration of 10 nM for 72 hours showed an increase in fluorescence with "shrunken" nuclei, characterized by the typical chromatin condensation (FIG. **10**B, indicated by arrows), which are peculiar to apoptotic cells. The results of the immunofluorescence assay were confirmed and quantified by using a specific ELISA test. In particular, FIG. **11**A shows a significant pro-apoptotic activity of the compound ML00253764 in A-2058 melanoma cells at the concentration of 10 nM (corresponding to the IC_{50}) after 72 hours of exposure, whereas using a 35 nM concentration (corresponding to the IC_{50}) of this compound induces a significant increase in the apoptotic signal in WM 266-4 cells after 72 hours of exposure (FIG. **11**B).

What is claimed is:

1. A method of therapeutic treatment of a tumor pathology selected from the group consisting of melanoma, tumors of the gastrointestinal tract, and thyroid carcinoma, said therapeutic treatment comprising administering to a subject in need thereof a melanocortin receptor-4 antagonist having formula (I)

(I)



or pharmaceutically acceptable salts and esters thereof, wherein

R and R^2 are independently a halogen atom; and

R^1 is a C_1-C_4 alkyl group.

2. The method of claim **1**, wherein R is a bromine atom and/or R^2 is a fluorine atom.

3. The method of claim **1**, wherein R^1 is a methyl group.

4. The method of claim **1**, wherein the melanocortin receptor-4 antagonist has formula (II)

(II)